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Imipramine's effect on REM sleep

Depression affects sleep, and sleep apparently is mediated by the same neurotransmitters found altered in depression. Consequently, the effects of antidepressants upon sleep are of special interest. Indeed, suppression in the rapid-eye-movement (REM) stage of sleep after imipramine overdose has been reported, and is followed by a rebound, though this has not been found with a single dose. In order to test the effects of a therapeutic dose of imipramine on REM sleep, 100 mg daily were given to 5 healthy young male subjects for 13 to 16 days. All-night sleep recordings were made before, during, and after this course of imipramine.

On the first drug night, REM sleep was either completely suppressed or markedly reduced. REM percent remained low during the entire drug period and increased sharply, but only to normal levels, on the first post-drug night. Not until post-drug night 5 or 6 was there a REM rebound peak of 50-100% above pre-drug levels, and REM percent did not return to normal levels before post-drug night 12 or 13. The difference between pre-drug mean REM percent (19.3%) and post-drug night 5 or 6 mean REM percent (31.5%) was significant with probability less than 0.005. Thus, the rebound peak found here, like that occurring after imipramine overdose, was delayed for several days after drug withdrawal; however it was shorted in duration. Consequently, therapeutic doses of imipramine cause REM suppression followed by a rebound qualitatively

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716. I.H. Jones, Melbourne, Victoria, Australia

An examination of the incidence of chromosomal gaps, breaks and hypodiploidy among psychiatric patients and non-psychiatric controls

During an examination by Dr. Garson of the effects of LSD on chromosomal structure in vivo it appeared that the incidence of chromosomal gaps, breaks and hypodiploidy might be greater in some psychiatric patients than controls irrespective of whether they had been given LSD or not.

The proposed paper describes an attempt to analyse various hypotheses derived from this initial observation, namely: (1) that chromosomal breaks and hypodiploidy are more common in psychiatric patients; (2) that these changes are more common in some groups of psychiatric patients than others; (3) that one or more of the chromosomal aberrations are the consequence of drug treatment; (4) that some psychoactive drugs are more liable to induce these effects than others.

This work is being carried out jointly by the Department of Medicine, St. Vincent's Hospital, and the Department of Psychiatry, University of Melbourne.

Psychiatric patients admitted to the University Psychiatric Unit are diagnosed according to precise psychiatric criteria. They are matched for age and sex with normal subjects or with surgical patients without a psychiatric history.

Chromosome studies are performed on admission on patients and controls. Further cytogenetic examinations are performed on patients and controls simultaneously before and after the patients are treated with psychoactive drugs. Sequential analysis and other statistical techniques are being used to examine the significance of the changes found.

The study is still in progress, statistically significant results have not yet been obtained but an association between a psychiatric diagnosis and hypodiploidy is approaching significance. Psychoactive drugs in general have not yet been shown by us to be significantly related to abnormal chromosomal configurations, differences in this respect between drugs are being examined.

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